STUDY REPORT

CANCER STATISTICAL REVIEW

for

West Point, Davis County, Utah

1975 - 2009

Environmental Epidemiology Program
Utah Department of Health
Salt Lake City, Utah

October 2012

TABLE OF CONTENTS

EXECUTIVE SUMMARY 3
INTRODUCTION 4
REQUEST AND STUDY AREA 5
DATA AND METHODS 5
FINDINGS 8
DISCUSSION 8
CONCLUSIONS AND RECOMMENDATIONS 15
AUTHORSHIP, ACKNOWLEDGMENT AND CITATION 16
CERTIFICATION 17
REFERENCES 18
FIGURE 1 30
TABLE 1 31
DEFINITIONS 37

CANCER STATISTICAL REVIEW

West Point, Davis County, Utah; 1975 - 2009

EXECUTIVE SUMMARY

In February 2012, the Environmental Epidemiology Program (EEP) received a request from Davis County Health Department (DCHD) to conduct a statistical review of cancer incidence in West Point, Davis County, Utah. DCHD reported that a resident had inquired about increased cancer cases occurring in a specific neighborhood in Davis County. DCHD did not provide EEP with any specific category of cancer or any indication of environmental concern related to the perception of increased cancer incidence in the neighborhood in question.

To conduct this investigation the EEP used a retrospective cross-sectional statistical review of cancer rates among residents within the study area defined by eight census block groups that included parts of the cities of Clinton, Clearfield, Syracuse and West Point. The 2000 census population was estimated to be 20,130 persons.

The investigation indicated that three digestive tract cancers were significantly elevated: esophageal, colon, and "other" cancers of the digestive tract. Each cancer elevation spanned only one five-year analytical period and none occurred concurrently. Esophageal cancer cases were elevated during the 2005-2009 analytical period. Though sufficient numbers of colon cancer cases existed to allow all analytical periods to be studied statistically, this cancer was significantly elevated only during the 1975-1979 period. Cancers classified as "other digestive tract" were elevated only during the 2000-2004 analytical period. The testicular cancer rate was elevated during the 1990-1994 period. Non-Hodgkin Lymphoma was elevated for the 1980-1984 period.

Risk factors for these cancers are discussed along with known environmental hazards near the study area.

CANCER STATISTICAL REVIEW
West Point, Davis County, Utah; 1975 - 2009

INTRODUCTION

What is Cancer? Cancer is a broad group of various diseases, all involving unregulated cellular division and tissue growth. Rapid cellular division and differentiation (the process whereby stem cells become a specific type of tissue) occur throughout fetal development and juvenile maturation. Once adulthood is achieved, some cells continue replication to replace damaged or dying cells. For example, the adult body replaces white blood cells every thirty days and red blood cells every four months. Stem cells divide and differentiate to replace the lost blood cells. In other tissues, cell division occurs to repair injuries, or when there is a need to build body mass (e.g., increasing muscle or fat mass, and skin surface area). Cell division is controlled by regulatory mechanisms at the DNA level. Cellular division can lead to non-functional growths when regulatory mechanisms are damaged or disrupted. These new growths are called neoplasms or more commonly called cysts, polyps or tumors. Some neoplasms may be benign. Benign tumors lack the ability to metastasize (spread to other parts of the body) and can usually be treated by removal. Other neoplasms may be pre-malignant or malignant cancers (possessing the ability to metastasize). Cancers are classified as "in-situ," meaning at the site of origin and not invading the surrounding tissue; "invasive," meaning at the site of origin and growing into (invading) surrounding tissues or "metastatic," meaning the cancer originating elsewhere in the body and migrating (usually through the blood or lymphatic systems) to a new location (ACS, 2012a; King and Robins, 2006; NCI, 2012a; Weinberg, 2006).

There are more than 100 different kinds of cancer, each possessing different physiological characteristics, causal risk factors, prognoses, and treatment (ACS, 2012a). When conducting a population-based statistical review, it is convenient to group similar cancers together, usually by location in the body. Cancer group definitions for major site-specific categories are those established by the Surveillance, Epidemiology and End Results (SEER) Program within the National Cancer Institute (NCI) and are used by the North American Association of Central Cancer Registries (NAACCR) (Adamo et al., 2011; Thorton, 2012). The use of these standardized classification schemes allows the comparison of the findings of this report with national and state cancer patterns (Copeland et al., 2011).

The Environmental Epidemiology Program (EEP), Utah Department of Health is occassionally asked by the public, either directly or through one of Utah's local health departments, to investigate a perceived excess of cancer cases in a neighborhood or area. The concern sometimes includes an environmental agent. These concerns are legitimate and responding to them is a responsible and empathetic public health practice (Bender et al., 1990; Caldwell, 1990; CDC, 1990; Kingsley et al., 2007; Thun and Sinks, 2004; Warner and Aldrich, 1988).

REQUEST AND STUDY AREA

Request: In February 2012, the EEP received a request from Davis County Health Department (DCHD) to conduct a statistical review of cancer incidence in West Point, Davis County, Utah. An individual had contacted DCHD about increased cancer in a specific neighborhood. DCHD did not provide EEP with any specific category of cancer or any indication of environmental concern related to the perception of increased cancer incidence in the neighborhood.

Study Area: The risk and incidence trends of cancer are difficult to assess at "neighborhood" levels. Part of the difficulty arises from the fact that this assessment is a statistical process that can be unstable and meaningless when working with small numbers (less than 30). Additionally, the legal requirements to protect confidentiality and privacy of the persons represented by case data imposes limitations on how EEP conducts an investigation and presents data. To overcome these challenges, the EEP defines a study population by aggregating several neighborhood areas until the included population is large enough to allow some temporal analysis while still maintaining statistical stability and protecting confidentiality.

Figure 1, presents a map of the study area defined by the aggregation of eight census block groups (49.011.125303.4, 49.011.124303.5, 49.011.125304.2, 49.011.125305.1, 49.011.125401.1, 49.011.125501.3, 49.011.125501.4, and 49.011.125503.5). The use of census block groups allows EEP to compute an incidence rate for a definable population. The estimated population for this study area based on the 2000 U.S. Census is 20,130 (Geolytics 2002c).

DATA AND METHODS

Study Design: This investigation is statistical review of cancer incidence rates among residents within the study area (Jekel et al., 1996; Mann, 2003). The incidences of cancer, quantified in five-year incidence rates of residents within the study area, were compared to corresponding incidence rates for the state of Utah. The study null hypothesis was that the incidence of cancer within the study area was not significantly different from the incidence of cancer for the state of Utah.

Cancer Data: Cancer incidence data on persons diagnosed with primary invasive cancers between 1975 and 2009 were obtained from the Utah Cancer Registry (UCR). This data includes diagnostic information, patient demographics, and residential addresses of the cases, as well as information about the behavior of the cancer. Individuals with multiple primary invasive cancers have multiple records in the data set in sequential order. These cancers are distinguished by individually unique cancer registry tracking numbers and a cancer sequence number. The sequence number allows discrimination between the very first cancer diagnosis and subsequent diagnoses (UCR, 2012). Diagnostic coding of cancers includes the International Classification of Disease Oncology, 3rd Edition (ICD-O-3) codes for site, histology and behavior (WHO, 2012). The UCR groups cancer into 42 major cancer types by site following the guidance provided by

the SEER Program (Adamo et al., 2011). These 42 UCR site codes are a convenient grouping for conducting surveillance analyses (UCR, 2012).

Cancer data were geo-coded (a process of indexing the street address to an x- and y-geographic coordinate) and geo-referenced (a process of using the x- and y-geographic coordinates to find the geographic small area in which the case belongs) by the EEP using ArcGIS version 10.0 (SP4). The EEP uses a composite address locator built on the statewide street feature layer provided by the Automated Geographic Reference Center (AGRC) and modified by EEP (AGRC, 2009). Geo-coding was conducted using standard methods and tools (EEP, 2009; McElroy et al., 2003). Geo-coded cancer data were geo-referenced to the Utah 2000 census block-group small areas (AGRC, 2002). Some error occurs in the geo-coding and geo-referencing process due to incomplete cancer address data, errors in the address locator data, or errors in the geographic representation of streets and census block-group features; however, these errors are thought to be non-systematic (Oliver et al., 2005; Ward et al., 2005; Zimmerman and Li, 2010).

When conducting an investigation of cancer risk associated with environmental concerns, it is necessary to exclude cancers that may have resulted from cancer treatment (sometimes known as therapy-induced cancer). Myeloid leukemia is one of these types of cancer (Godley and Larson, 2008; Leone et al., 1999; Leone et al., 2011; Sill et al., 2011; Wilkins and Woodgate, 2008). The UCR indicated that among the study area residents, 1,076 incidents of cancer were diagnosed in 965 individuals between 1975 and 2009. Fourteen cases of carcinoma in-situ (pre-invasive cancer) and three cases of non-first sequence primary myeloid leukemia were excluded from analysis because cancer treatment is a risk factor for subsequent primary leukemia; therefore, statistical assessment included 1,059 cases of invasive primary cancer cases.

Population Data: The 2000 U.S. census divides Utah into 1,481 census block groups (USCB, 2004). Commercially available U.S. census population data for Utah for the 1970, 1980, 1990, 2000 and 2010 censuses were obtained from Geolytics, Inc. (Geolytics, 2002 a-c; Geolytics 2012 a-b). Geolytics provided the 1980, 1990, 2000, and 2010 data allocated into the 2000 census boundary schema. The EEP used the census block group to county ratio for the 1980 census to allocate the 1970 population counts into the year 2000 census boundary areas. Intercensal population estimates were made by linear extrapolation between the census years and projected to 2019 using the same slope derived for the 2001 to 2009 estimation.

Indirect Age-Standardized Incidence Rates: SAS version 9.2 (a statistical analyses program) was used to manage and analyze the data. Cancer case and population data were aggregated into six age cohorts: 0-19 years of age, 20-34 years of age, 35-49 years of age, 50-64 years of age, 65-74 years of age, and 75 years and older. The indirect age-standardized incidence rate was calculated using standard methods (Anderson and Rosenberg, 1998; Jekel et al., 1996; Klein and Schoenborn, 2001; Lawson and Williams, 2001; Selvin, 1996; Waller and Gotway, 2004). The five-year estimated population for the state of Utah was centered on the year 2000 (1998 - 2002)

and used as the "standard population" for this study (Klein and Schoenborn, 2001; Shalala, 1998). All Utah cancer cases falling within the same time period as the standard population were selected using the same exclusion criteria as the study population. This information was used to generate a standard rate of cancer. The indirect standardization method is preferred when the disease being investigated is rare (Lawson and Williams, 2001; Waller and Gotway, 2004).

For sex-specific cancers (e.g., cancers of the breast, cervix, uterus, ovary, other female genital, prostate, testis, or other male genital) only the population counts of the appropriate sex were used. All occurrences (first and subsequent) of primary cancer incidence were used except for leukemia. As described above, the treatment of other types of cancer (i.e., chemotherapy and radiotherapy) increase the risk of incidence of myeloid leukemia (Godley and Larson, 2008; Leone et al., 1999; Leone et al., 2011; Sill et al., 2011; Wilkins and Woodgate, 2008). Therefore, individuals with these cancers were considered cases only if the incidence of these cancers was the first incidence (first in the sequence). This study does not consider pre-cancer (e.g., carcinoma in-situ) or non-primary cancers (e.g., cancers arising due to metastasis of a primary cancer).

Indirect age-standardized incidence rates were computed for the incidence for each of seven 5-year analytical periods (e.g., 1975-1979, 1980-1984, 1985-1989, 1990-1994, 1995-1999, 2000-2004, 2005-2009).

Standardized Incident Rate Ratio: The standardized incidence rate of cancer for the study population is evaluated against the incidence rate of cancer for the state of Utah using the standardized incidence rate ratio (SIR). An SIR greater than one (1.0) indicates that the cancer incidence rate of the study population is greater than the state's for that period of analysis. Conversely, an SIR less than one indicates that the study population cancer incidence rate is less than the state's rate. Statistical significance is determined by applying the Byar's 95% confidence interval for the SIR (Berslow and Day, 1987; Rothman and Boice, 1979, 1982; Sahai and Khurshid, 1983, 1996). The criteria for a specific cancer to demonstrate statistical significance is an SIR greater than one (1.0) and a confidence interval (expressed by the lower and upper limits) that does not include one (1.0).

For statistical validity, SIRs and corresponding 95% confidence intervals were only calculated for time periods with four or more cases (Bender et al., 1990; Caldwell, 1990; Thun and Sinks, 2004). Using a 95% confidence interval is a well-established standard for interpretation of an SIR with respect to statistical significance. It should be noted that an SIR may be significant using this interpretation criteria, yet simply be a mathematical artifact without biological meaning or relevance (Bender et al., 1990; Besag and Newell, 1991). When conducting multiple analyses using the 95% confidence interval to interpret the data, one would expect one in twenty (5%) of the analyses to have a significant interpretation as a result of random chance. The EEP considers the results meaningful if there are two consecutive time periods with a significant result, and the number of cases in each time period is greater than three (Langeberg et al., 2004).

The EEP is required to protect confidential data from unlawful disclosure; therefore, the EEP does not report the exact results for time periods containing three or less cases.

FINDINGS

The standardized incidence rates and SIR were calculated for each analytical cell (defined as the study area by the seven 5-year study periods and by the 42 cancer site categories defined by the UCR) that had at least three incidences of cancer. Table 1 presents data for those analytical cells for which a standardized incidence rate and SIR were calculated. All other analytical cells did not have at least three incidences of cancer and are suppressed.

Identification of cancer types that are statistically elevated in Table 1 is based on the SIR and the 95% confidence limit to the SIR (presented in parentheses following the SIR value). If the SIR is greater than 1.0 and the lower confidence limit does not include 1.0 (confidence is greater than 1.0) then the rate is interpreted to be significantly elevated. If there are two consecutive analytical periods of statistically elevated rates, then the rates are considered meaningful as well. No cancer site categories in this study met these requirements. Therefore, no cancer clusters were indicated.

The investigation indicated that three digestive tract cancers were significantly elevated: esophageal, colon, and "other" cancers of the digestive tract. Each cancer elevation spanned only one five-year analytical period and none occurred concurrently. Esophageal cancer cases were elevated during the 2005-2009 analytical period. Though sufficient numbers of colon cancer cases existed to allow all analytical periods to be studied statistically, this cancer was significantly elevated only during the 1975-1979 period. Cancers classified as "other digestive tract" were elevated only during the 2000-2004 analytical period. The testicular cancer rate was elevated during the 1990-1994 period. Non-Hodgkin Lymphoma was elevated for the 1980-1984 period.

DISCUSSION

Cancer: Cancer is a broad group of more than 100 diseases that involve uncontrollable cell replication and growth. Often these cells are "undifferentiated" meaning they have lost their tissue specific characteristics. As these cells grow to form tumor tissue, they invade nearby healthy tissue or spread through metastasis to other tissues. This invasion, or spread, disrupts the functions of the affected healthy tissues. Cancer cells may also produce metabolic products that can be transported to other parts of the body resulting in adverse health effects (ACS, 2012a; NCI, 2012a). The American Cancer Society (ACS) estimates that about one in two men and one in three women will develop cancer (all invasive sites) sometime in their life (ACS, 2012b; NCI, 2011a-b). In the United States, cancer is the second leading cause of death (CDC, 2012). Among all causes of death, approximately, one in four men and one in five women will die of cancer

(ACS, 2012b; NCI, 2011a-b). On average, about one in nine people will develop two or more cancers in his or her lifetime (Wilkins and Woodgate, 2008).

Risk factors that contribute to the development of cancer include both inherent and external factors. Inherent factors include a variety of genetic susceptibilities. External factors include life choices and behaviors (e.g., tobacco use, alcohol use, poor diet, obesity, lack of physical activity, excessive sunlight exposure, and sexual behavior), medical conditions and medications, oncogenic pathogens, and chemical or radiological environmental exposures. Cancer may be the result of several factors interacting together with a triggering event (ACS, 2012, NCI, 2012a-b).

Cancer of the esophagus: There are two kinds of esophageal cancer; squamous cell carcinoma and adenocarcinoma. Adenocarcinoma arises from the glandular cells that are present at the junction of esophagus and the stomach (NCI, 2012b). Squamous cell carcinoma arises from the cells that line the esophagus. This type of cancer is usually found in the upper part of the esophagus. Around the world this has historically been the most common form of esophageal cancer. In the United States, prior to the 1960's, 90% of esophageal cancers were squamous cell carcinomas. Today, the incidence of adenocarcinoma and squamous cell carcinoma is almost equal (Gallo and Cha, 2006).

Esophageal cancer is both common and deadly (Gallo and Cha, 2006). Worldwide esophageal cancer is the 8th most common incident cancer and because of its high fatality rate, ranks 6th among all cancers in mortality (Ferlay et al., 2010; Kamangar et al., 2006; Parkin et al., 2006). Since 1980, the incidence and mortality of esophageal cancer in the United States and in Utah has steadily increased. Utah rates have remained lower than the U.S. rates. In 2008, the Utah incidence rate was 3.5 new cases per 100,000 persons, and the mortality rate was 2.7 deaths per 100,000 persons. Davis County, Utah, has the highest incidence rate for esophageal cancer of any of Utah's counties (NCI, 2012c).

There are a number of risk factors associated with the development of esophageal cancer. Most esophageal cancers can unequivocally be linked to cigarette smoking (Kamangar et al., 2009; Kollorova et al., 2007; Layke and Lopez, 2006; Morita et al., 2010; Thun et al., 2002; Toh et al., 2010). Weight, weight gain, obesity and conditions associated with obesity account for approximately 20-25% of all cancer cases and 37% of esophageal cancer (Guh et al., 2009; Wolin et al., 2010).

Another risk factor, gastroesophageal reflux disease (GERD), is a condition where acidic stomach content chronically leaks into and irritates or damages the esophagus. GERD can lead to the development of Barrett's esophagus where the function and morphology of the cells lining the esophagus change (called metaplasia). The normal squamous cells are replaced by columnar cells. GERD and Barrett's esophagus have been shown to be significant risk factors for development of esophageal adenocarcinoma (Cossentino and Wong, 2003; Fennerty, 2003; McManus et al., 2004; Shaheen and Ransohoff, 2002; Von Rahden et al., 2003; Zhang et al.,

2009). Another disorder, associated with GERD or a hiatal hernia, is achalasia, an esophageal disorder in which the lower esophageal sphincter has difficulty relaxing. Achalasia is a known risk factor for esophageal cancer (Kamangar et al., 2009).

Interestingly, the use of stomach acid inhibitory medications is associated with the development of esophageal cancer. Acid inhibitory medications are protective in that they reduce acid reflux and esophageal cancer; however, they also change the pH of the stomach and microbial flora which can result in an increase in the production of potentially harmful compounds by these organisms (Duan et al., 2009; Garcia-Rodriquez et al., 2006; Layke and Lopez, 2006).

Excessive consumption of certain pickled vegetables, alcohol, yerba mate (an herbal tea), and hot foods and drinks are indicated as risk factors for esophageal cancer (Brooks et al., 2009; Kamangar et al., 2009; Kollorova et al., 2007; Layke and Lopez, 2006; Morita et al., 2010; Toh et al., 2010). Alcohol is thought to damage the esophageal lining necessitating cellular replacement (i.e. healing). The healing process increases the risk of cell division errors, thus giving rise to cancer. The International Agency for Research on Cancer (IARC, 2012) has classified certain kinds of Asian pickled vegetables and yerba mate as potentially carcinogenic to humans (Group 2B and Group 2A, respectively) based on the presence of carcinogenic compounds in these products and their potential to physically damage cells lining the esophagus.

Other potential risk factors include low intake of fresh fruits and vegetables, selenium and zinc deficiencies, low socioeconomic status, infection with the human papillomavirus, and physical and chemical injuries (e.g., ionizing radiation, traumatic burns, etc.) (Gonzalez et al., 2006; Kamangar et al., 2009; Kollorova et al., 2007; Riboli and Norat, 2003). Environmental exposure risk factors include certain polycyclic aromatic hydrocarbons, n-nitroso compounds, acetaldehyde, fumonisins (toxins secreted by the fungus *Fusarium verticilliodes)*, exposure to asbestos, and petroleum vapors and combustion bi-products (Jakszyn and Gonzalez, 2006; Kamangar et al., 2009; Kollorova et al., 2007).

Cancer of the colon: In 2012, the NCI estimates that more than 143,000 people in the United States will be diagnosed with colorectal cancer, making this type of cancer the fourth most common cancer in men (following skin, prostate and lung cancer) and in women (following skin, breast and lung cancer) (NCI, 2012b). In Utah, the incidence rate for colorectal cancer between 2004 and 2007 was 36.3 cases of cancer per 100,000 persons, compared to the U.S. incidence rate of 47.6 for the same period. The incidence is highest in Sanpete, Sevier, Carbon and Grand counties. These counties have rates higher than the U.S. rate. The mortality rate in Utah is 12.2 deaths per 100,000 persons compared the U.S. mortality rate of 17.1 deaths per 100,000 persons. Both the incidence and mortality rate of colorectal cancer has been declining in recent years (NCI, 2012c).

Most risk factors for colorectal cancer result from controllable behaviors and life choices. Tobacco use, particularly smoking, increases the risk for developing colorectal cancer by two-to-

three times (Botteri et al., 2008; Giovannucci, 2001, Liang et al., 2009). Excessive alcohol consumption is also a major risk factor for colorectal cancer (Giovannucci, 2004).

Weight gain and obesity are the most important contributors to cancer risk of all types including colorectal cancer (Freeza et al., 2006; Guh et al., 2009; Larsson and Wolk, 2007; Pischon et al., 2006; Wolin et al., 2010). Associated with weight control is diet. High fat diets, and diets that are low in calcium, folate, or fiber increase the risk of colorectal cancer (Aune et al., 2011; Campos et al., 2005; Dai et al., 2007; Du et al, 2010; Duthie et al., 2004; Giovannucci, 2002a-b, Giovannucci, 2003; Moghaddam et al., 2007). Vitamin D deficiency has also been associated with increased risk (Davis, 2008; Davis and Milner, 2011). Consumption of excessive fat or sugar results in hyperinsulinemia (high blood insulin levels). Insulin has an effect on promoting cell death and cell growth. Hyperinsulinemia may be one of the mechanisms that diet has on the risk for colorectal cancer (Giovannucci, 2007).

Family history of colorectal cancer is an indicator for increased personal risk for developing this cancer. In addition, a personal history of prior cancer increases one's risk for the development of colorectal cancer. Familial risk is thought to be associated with several different genetic alterations (Ahnen, 1991; Ballinger and Anggiansah, 2007; Calvert and Frucht, 2002; Dunlop 1992; Gatalica and Torlakovic, 2008; Jasperson et al., 2010; Rustgi 2007; Vasen et al. 2009).

Closely associated with genetic risk are two conditions that can develop into cancer. Colorectal polyps are growths on the inner wall of the colon and rectum. They are common in people over age 50. Most polyps are benign, but some (adenomas) can become cancer. Finding and removing polyps by regular checkup may reduce the risk of colorectal cancer. Some persons may have a genetic predisposition to developing colorectal adenomas (Half et al., 2009; Neugut et al., 1993; Read and Kodner, 1999). Both men and women over the age of 50 years should be screened on a regular basis for colorectal polyps. Another condition that may lead to the development of colorectal cancer is ulcerative colitis or Crohn's disease (Ballinger and Anggiansah, 2007; Lakatos and Lakatos, 2008; Xie and Itzkowitz, 2008; Zisman and Rubin, 2008).

Other digestive tract cancers: These are primarily cancers of the peritoneum (a thin, delicate sheet of connective tissue that lines the inside wall of the abdominal and pelvic cavity), the omentum (a fold in the peritoneum that provides a fatty apron like protective cushion for the organs in abdominal cavity), the mesentery (the membranous tissues that connect to and enclose the organs in the abdominal and pelvic cavity and anchor them in place), the retroperitoneum (the space between the abdominal wall and the peritoneum), and the structures associated with these tissues (Patton and Thibodeau, 2000; UCR, 2012).

A variety of types of primary cancer can arise in these soft tissues (Mack, 1995). Primary cancers of these tissues are rare and risks associated with these cancers are not well developed. Some primary cancers arising in these tissues are thought to be linked to genetic mutations or genetic dysfunction (Casey and Bewtra, 2004; Grant et al., 2010; Menczer et al., 2003). Weight, weight

gain, and obesity are additional risk factors (Grant et al., 2010; Guh et al., 2009; Wolin, et. Al., 2010). Exposure to asbestos is a known risk factor for the pleura and peritoneum of thoracic cavity and may be a risk factor for the abdominal mesothelioma of the peritoneum as well (BTS-SCC, 2001, 2007; Clin et al., 2009; De La Provote et al., 2002; Jarvholm and Sanden, 1998). Often cancers of these tissues are associated with or diagnosed to one of the organs contained within the peritoneum (including organs in other systems, besides the digestive tract) (Goodman and Shvetsov, 2009; Grant et al., 2010).

Testicular cancer: Testicular cancer is the most common neoplastic malignancy in 20- to 34-year-old males and the lifetime risk, which is approximately 0.5–1%, has increased nearly three-fold in the last five decades (Huyghe et al., 2003; Kinkade, 1999). More than 95% of testicular tumors are of germ cell origin [testicular germ cell tumors (TGCTs)] and can be divided into two main types: seminomas and non-seminomas. The latter type may consist of several components, including embryonal carcinoma, teratoma, polyembryoma, and choriocarcinoma or yolk sac tumor. In approximately 10% of cases both seminoma and non-seminoma may develop simultaneously in one testicle as the so-called combined (or mixed) germ cell tumor (Kinkade, 1999; Hoei-Hansen et al., 2005).

The risk factors for testicular cancer are not well substantiated. Race, particularly Caucasian race, is the only consistent risk factor. Testicular cancer is five times more common in white than black men, and three times more common in white than in Asian or Native American men. Body height is also positively associated with increased risk for testicular cancer. Various medical conditions such as cryptorchidism (undescended testicles), impaired spermatogenessis (production of sperm cells), inguinal hernia, hydrocele (accumulation of excess fluid in the scrotom), genetic developmental disorders (e.g., Klinefelter's syndrome), atopy (genetic predisposition to hypersensensitivity and allergic reactions), and testicular atrophy (shrinkage) are thought to be the most important risk factors. In addition, certain kinds of viral factors have been associated with the development of testicular cancer. These include Human Immuno-deficiency Virus (HIV), Epstein-Barr virus and Cytomegalovirus infections. Injury to the testes, including those due to medical procedures (such as a prior testicular biopsy), may also contribute to risk. There is some evidence of a familial genetic predisposition. Prenatal risk such as low birth weight or pre-term birth, maternal smoking and use of diethystilbestrol may be risk factors. Investigations into these risk factors have yielded mixed results. Environmental exposures to certain kinds of endocrine-disrupting chemicals have been postulated as a risk (Kinkade, 1999; Kratz et al., 2010; Manecksha et al., 2009; McGlynn and Cook, 2009; O'Callaghan and Mead, 1997; Schager and Potter, 2004).

Non-Hodgkin Lymphoma: Non-Hodgkin lymphoma is a type of lymphoma, a cancer that starts in white blood cells called lymphocytes. Lymphocytes are part of the body's immune system. There are two kinds of lymphomas: Hodgkin lymphoma (named after Dr. Thomas Hodgkin, who recognized it in 1832), and non-Hodgkin lymphoma. These two main types of lymphomas differ in how they behave, spread, and respond to treatment. Lymphomas are cancers that arise in

lymphocytes. Lymphocytes are a type of white blood cell that helps the body fight infections. There are two major types of lymphocytes, known as B-cell lymphocytes and T-cell lymphocytes. T-cell lymphocytes are involved in producing substances that help other kinds of white blood cells fight infections or respond to injuries. B-cell lymphocytes make antibodies against germs. Lymphocytes are found throughout the body, but tend to collect in certain kinds of tissues including the lymph nodes (hence their name), spleen, bone marrow, and thymus. Lymphoma can start anywhere lymphocytes are found, but most often start in the lymph nodes in the upper part of the body. Lymphoma occurs when lymphocytes are produced in an out-of-control excessive rate (ACS, 2012c; NCI, 2012b).

They are many types of non-Hodgkin lymphoma and classifying them (even for doctors) can be confusing. Non-Hodgkin lymphoma can start with either B-cell or the different kinds of T-cell lymphocytes. Overall, the risk of non-Hodgkin lymphoma is higher in men than in women, but there are certain types of non-Hodgkin lymphoma that are more common in women. In the United States, whites are more likely than African Americans and Asian Americans to develop non-Hodgkin lymphoma. Non-Hodgkin lymphoma is more common in older people (ACS, 2012c).

People with weakened immune systems have an increased risk for non-Hodgkin lymphoma. For example, people who receive organ transplants (kidney, heart, liver) are treated with drugs that suppress their immune system to prevent it from attacking the new organ. These people have a higher risk of developing non-Hodgkin lymphoma. The human immunodeficiency virus (HIV) can also weaken the immune system, and people infected with HIV are at increased risk of non-Hodgkin lymphoma. Some genetic (inherited) syndromes can cause children to be born with a deficient immune system. Along with an increased risk of serious infections, these children also have a higher risk of developing non-Hodgkin lymphoma. These inherited immune deficiency diseases can be passed on to children, but people with non-Hodgkin lymphoma who do not have these inherited diseases do not pass an increased risk of lymphoma on to their children (ACS, 2012c; NCI, 2012b).

Some autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus (SLE, or lupus), celiac sprue (gluten-sensitive enteropathy), and others have been linked with an increased rate of non-Hodgkin lymphoma. In autoimmune diseases, the immune system sees the body's own tissues as foreign and attacks them, as it would a germ. Lymphocytes (the cells from which lymphomas start) are part of the body's immune system. The overactive immune system in autoimmune diseases may cause lymphocytes to grow and divide more often than normal. This may increase the risk of them developing into lymphoma cells (ACS, 2012c).

In addition to HIV infections, other types of infections, such as human T-cell leukemia/lymphoma virus (HTLV-1) and the Epstein-Barr virus (EBV) may raise the risk of non-Hodgkin lymphoma. These viruses infect lymphocytes and can directly affect the DNA of infected cell, helping to transform them into cancer cells. Almost all people living in the United

States have been infected by EBV (the cause of mononucleosis), usually in their early childhood (ACS, 2012c; NCI, 2012b).

Exposure to chemicals such as benzene and certain herbicides and insecticides (weed- and insect-killing substances) may be linked with an increased risk of non-Hodgkin lymphoma. Some chemotherapy drugs used to treat other cancers may increase the risk of developing non-Hodgkin lymphoma many years later. For example, patients who have been treated for Hodgkin lymphoma have an increased risk of later developing non-Hodgkin lymphoma. Human herpes virus 8 (HHV8), hepatitis C virus (HCV) and *Helicobacter pylori* are also known to increase the risk for developing non-Hodgkin lymphoma (ACS, 2012c).

Studies of survivors of atomic bombs and nuclear reactor accidents have shown they have an increased risk of developing several types of cancer, including leukemia, thyroid cancer, and non-Hodgkin lymphoma. Patients treated with radiation therapy for some other cancers, such as Hodgkin lymphoma, have a slightly increased risk of developing non-Hodgkin lymphoma later in life. This risk is greater for patients treated with both radiation therapy and chemotherapy (ACS, 2012c).

Some studies have suggested that being overweight or obese may increase your risk of non-Hodgkin lymphoma. Other studies have suggested that a diet high in fat and meats may raise your risk. More research is needed to confirm these findings (ACS, 2012c).

Potential environmental risk factors in West Point, Syracuse, and Clinton: No industrial environmental risks were identified within the study area. Eight Toxic Release Inventory (TRI) sites, all associated with the Freeport Center in Clearfield are found within a mile of the study area. TRI sites are industries that emit nationally listed hazardous pollutants into the air, water, or ground at a reportable level. The TRI sites currently or previously located in the Freeport Center include Allied Signal, Ashland Distribution, Futura Industries, Lifetime Products, NapTech, NapTech Pressure Systems, and WR Grace Construction. In 2010, Ashland Distribution released 3,365 pounds of organic solvents (diethanolamine, certain glycol ethers, methanol, n-hexane, styrene, toluene, 1,2,4-trimethylbenzene, and xylene); Futura Industries released 22 pounds of nitric acid; Lifetime Products released 15 pounds of metals (chromium, manganese and nickel); and WR Grace Construction released 168 pounds of nitrate compounds. The other sites did not report in 2010 and are no longer operating (USEPA, 2012a).

Two sites listed in the "Comprehensive Environmental Response, Compensation, and Liability Information System" (CERLIS) are also located in the Freeport Center. CERCLIS sites are contaminated with hazardous substances and are in the process of be prioritized for clean-up through the "Superfund" program. These two CERCLIS sites located in Freeport Center are the abandoned Freeport Center Naval Supply Depot and North American Environmental facilities (USEPA, 2012b).

CONCLUSIONS AND RECOMMENDATIONS

Cancer incidence rates were evaluated and grouped by the 42 SEER site categories. The EEP requires at least two consecutive analytical periods to have statistically elevated results to determine the incidence of a particular type of cancer is elevated. The EEP requires that a cancer site category have least two consecutive analytical periods to have statistically elevated incidence rates, before the EEP can determine that type of cancer to be a cluster. No site category met the criteria; however, cancer incidence were found to be elevated for one analytical period for esophageal, colon and other digestive tract cancers, testicular cancer and non-Hodgkin lymphoma.

An investigation of these cancer categories found that unhealthy lifestyle and life choices (e.g., tobacco use, alcohol use, excessive weight and obesity, unhealthy diet) contributed the greatest risk. These are controllable risk factors. Certain kinds of mutations and other genetic conditions may result in increased risk. Some types of medical conditions and medical procedures may also increase the risk for cancer. Environmental exposures to certain harmful chemicals may also increase the risk for cancer development. Genetics, medical conditions and environmental exposures are uncontrollable factors.

The EEP recommends that DCHD coordinate with EEP and the Utah Cancer Control Program (see http://cancerutah.org, 1-800-717-1811) to organize and provide cancer health education to the concerned community. Education can include a presentation and discussion of this report, recommendations for limiting controllable risk, and resources available for screening, prevention, early detection, and treatment. This training is particularly important for colon cancer. Screening for these cancers should be part of routine health care maintenance.

While no cancer category met the required two consecutive analytical periods with elevated results, the statistical results do suggest that a follow-up investigation in three to five years is warranted. The DCHD should consider requesting a follow-up investigation for this community in the future when additional data is available.

CANCER STATISTICAL REVIEW

West Point, Davis County, Utah; 1975 - 2009

Prepared by

Sam LeFevre
Environmental Epidemiology Program Manager

Craig Dietrich, PhD
Toxicologist and Environmental Health Hazards Assessment Team Manager

Acknowledgments

Cancer data used for this investigation was obtained from the Utah Cancer Registry. The Utah Cancer Registry is funded by contract N01-PC-35141 from the National Cancer Institute's SEER Program with additional support from the Utah Department of Health and University of Utah.

Other data and analytical tools used for this investigation were obtained from the Utah Environmental Public Health Tracking Network. In addition, the Utah Environmental Public Health Tracking Network provides geocoding services to Utah Cancer Registry data. The Utah Environmental Public Health Tracking Network is funded by a grant from the Centers for Disease Control and Prevention, Environmental Public Health Tracking Branch. The current Utah Environmental Public Health Tracking Network award is number 1U38EH000954.

Suggested citation

Environmental Epidemiology Program (EEP). Statistical Cancer Review for West Point, Davis County, Utah from 1975 through 2009. Salt Lake City, Utah: Utah Department of Health, March 2012.

Copyright information

CERTIFICATION

This report titled "Cancer Statistical Review: West Point, Davis County, Utah; 1975 to 2009" was prepared by the Environmental Epidemiology Program, Utah Department of Health. This report covers an investigation of cancer incidence using standard and approved methodology and procedures existing at the time the investigation herein reported was begun. Editorial and technical review was completed by UDOH certifying reviewers and program partners.

Approved by:

Sam LeFevre, Manager
Environmental Epidemiology Program
Utah Department of Health

Jennifer Brown, JD, Director
Bureau of Epidemiology
Utah Department of Health

Barry Nangle, PhD, Manager
Center for Health Data
Utah Department of Health

REFERENCES

Web links provided as part of a reference to a government or organizational website may wrap to multiple lines.

Adamo MB, Johnson CH, Ruhl JL, Dickie LA, eds. 2011 SEER program coding and staging manual. NIH publication 11-5581. Bethesda, MD: National Cancer Institute. January 2011. (See Appendix C - Site specific coding modules). Available at: http://seer.cancer.gov/tools/codingmanuals/index.html on March 27, 2012.

Ahnen DJ. Genetics of colon cancer. *Western Journal of Medicine*. 1991; 154(6):700-705.

American Cancer Society (ACS). What is cancer? website. Atlanta, GA: American Cancer Society.2012a. Available at: http://www.cancer.org/Cancer/CancerBasics/what-is-cancer on March 26, 2012a.

American Cancer Society (ACS). Lifetime risk of developing or dying from cancer web site. Atlanta, GA: American Cancer Society. 2012b. Available at: http://www.cancer.org/Cancer/CancerBasics/lifetime-probability-of-developing- or-dying-from-cancer on March 26, 2012b.

American Cancer Society (ACS). Non-Hodgkin Lymhoma. Atlanta, GA: American Cancer Society. 2012c. Available at: http://www.cancer.org/acs/groups/cid/documents/webcontent/003126-pdf.pdf on August 27, 2012.

Anderson RN, Rosenberg HM. Age standardization for death rates: implementation of the year 2000 standard. *National Vital Statistics Report*. 1998; 47(3):1-17.

Aune D, Chan DS, Lau R, Vieira R, Greenwood DC, Kampman E, Norat T. Dietary fibre, whole grains, and risk of colorectal cancer: systematic review and dose-response meta-analysis of prospective studies. BMJ. 2011; 243:d6617.

Automated Geographic Reference Center (AGRC). SGID93_Demographic_ CensusBlocGroups2000 shape file, computer data file from the State Geographic Information Database. Salt Lake City, UT: Department of Information Technology. 2002.

Automated Geographic Reference Center (AGRC). SGID93_Transportation_RoadsTIGER2009 shape file, computer data file from the State Geographic Information Database. Salt Lake City, UT: Department of Information Technology. 2009.

Ballinger AB, Anggiansah C. Colorectal cancer. *BMJ*. 2007; 335(7622):715-718.

Bender AP, Williams AN, Johnson RA, Jagger HG. Appropriate public health response to clusters: the art of being responsibly responsive. *American Journal of Epidemiology*. 1990; 132(Suupl 1):S48-S52.

Berslow NE, Day NE. Statistical methods in cancer research. Vol. II, the design and analysis of cohort studies (IARC Scientific Publication No 82). Lyon, France: International Agency for Research on Cancer. 1987.

Besag J, Newell J. The detection of clusters of rare disease. *Journal of the Royal Statistical Society, Part A*. 1991; 154:143-155.

Botteri E, Iodice S, Bagnardi V, Raimondi S, Lowenfels AB, Maisonneuve P. Smoking and colorectal cancer: a meta-analysis. *JAMA*. 200; 300(23):2765-2778.

British Thoracic Society Standards of Care Committee (BTS-SCC). Statement on malignant mesothelioma in the United Kingdom. *Thorax*. 2001; 56(4):250-265.

British Thoracic Society Standards of Care Committee (BTS-SCC). BTS statement on malignant mesothelioma in the UK, 2007. *Thorax*. 2007; 62(Suppl 2):ii1-ii19.

Brooks PH, Enoch MA, Goldman D, Li TK, Yokoyama A. The alcohol flushing response: an unrecognized risk factor for esophageal cancer from alcohol consumption. *PloS Medicine*. 2009; 6(3):e50.

Caldwell GG. Twenty-two years of cancer cluster investigations at the Centers for Disease Control. *American Journal of Epidemiology*. 1990; 132(Suppl 1):S43-S47.

Calvert PM, Frucht H. The genetics of colorectal cancer. *Annals of Internal Medicine*. 2002; 137(7):603-612.

Campos FG, Logullo Waitzberg AG, Kiss DR, Waitzberg DL, Habr-Gama A, Gama-Rodrigues J. Diet and colorectal cancer: current evidence for etiology and prevention. Nutrición Hospitalaria. 2005; 20(1):18-25.

Casey MJ, Bewtra C. Peritoneal carcinoma in women with genetic susceptibility: implications for Jewish populations. *Familial Cancer*. 2004; 3(3-4):265–281.

Centers for Disease Control and Prevention (CDC). Guidelines for investigating clusters of health events. *Morbidity and Mortality Weekly Report: Recommendations and Reports*. July 27, 1990; 39(RR-11):1-16.

Centers for Disease Control and Prevention (CDC). Leading causes of death web page. Centers for Disease Control and Prevention: Atlanta, Georgia. 2012. Available at: http://www.cdc.gov/nchs/fastats/lcod.htm on March 26, 2012.

Clin B, Morlais F, Dubois B, Guizard AV, Desoubeaux N, Marquignon MF, Raffaelli C, Paris C, Galateau-Salle F, Launoy G, Letourneux M. Occupational asbestos exposure and digestive cancers - a cohort study. *Alimentary Pharmacology and Therapeutics*. 2009; 30(4):364-374.

Copeland G, Lake A, Firth R, Xiao-Cheng W, Stroup A, Russell C, Boyuk K, Niu X, Schymura MJ, Hofferkamp J, Kohler B, *eds*. Cancer in North America: 2004-2008; Vol 1: combined cancer incidence for the United States and Canada. Springfield, Ill.: North American Association of Central Cancer Registries. May 2011. Available at: http://www.naaccr.org/ DataandPublications/CINAPubs.aspx on March 27, 2012

Cossentino MJ, Wong RK. Barrett's esophagus and risk of esophageal adenocarcinoma. *Seminars in Gastrointestinal Disease*. 2003; 14(3):128-135.

Dai Z, Xu YC, Niu L. Obesity and colorectal cancer risk: a meta-analysis of cohort studies. *World Journal of Gastroenterology*. 2007; 13(31):4199-4206.

Davis CD. Vitamin D and cancer: current dilemmas and future research needs. *American Journal of Clinical Nutrition*. 2008; 88(2):565S-569S.

Davis CD, Milner JA. Vitamin D and colon cancer. *Expert Reviews on Gastroenterology and Hepatology*. 2011; 5(1):67-81.

de La Provôté S, Desoubeaux N, Paris C, Letourneux M, Raffaelli C, Galateau-Salle F, Gignoux M, Launoy G. Incidence of digestive cancers and occupational exposure to asbestos. European Journal of Cancer Prevention. 2002; 11(6):523-528.

Du W, Li WY, Lu R, Fang JY. Folate and fiber in the prevention of colorectal cancer: between shadows and the light. *World Journal of Gastroenterology*. 2010; 16(8):921-926.

Duan L, Wu AH Sullivan-Halley J, Bernstein L. Antacid drug use and risk of esophageal and gastric adenocarcinomas in Los Angeles County. *Cancer Epidemiology, Biomarkers and Prevention*. 2009; 18(2):526-533.

Dunlop MG. Colorectal cancer genetics. *Seminars in Cancer Biology*. 1992; 3(3):131-140.

Duthie SJ, Narayanan S, Sharp L, Little J, Basten G, Powers H. Folate, DNA stability and colo-rectal neoplasia. *Proceedings of the Nutrition Society*. 2004; 63(4):571-578.

Environmental Epidemiology Program (EEP). Geocoding and georeferencing standard operating procedure. Salt Lake City, UT: Utah Department of Health. 2009.

Fennerty MB. The continuum of GERD complications. *Cleveland Clinical Journal of Medicine*. 2003; 70(Suppl 5):S33-S50.

Ferlay J Shin H-R, Bray F, Forman D, Mathers C, Parkin DM. Estimates of worldwide burden of cancer in 2008: GLOBOCAN 2008. *International Journal of Cancer*. 2010; 127(12):2893-2917.

Frezza EE, Wachtel MS, Chiriva-Internati M. Influence of obesity on the risk of developing colon cancer. *Gut*. 2006; (55(2):285-291.

Gallo A, Cha C. Updates on esophageal and gastric cancers. *World Journal of Gastroenterology*. 2006; 12(20):3237-3242.

Garcia-Rodriguez LA, LagergrenJ, Lindbald M. Gastric acid supression and risk of oesophageal and gastic adenocarcinoma: a nexted case control study in the UK. *Gut*. 2006; 55(11):1538-1544.

Gatalica Z, Torlakovic E. Pathology of the hereditary colorectal carcinoma. *Familial Cancer*. 2008; 7(1):15-26.

Geolytic, Inc. Census CD 1970, Release 2.0 on digital optical disk (CD). 2002a. Available at: http://www.GeoLytics.com. Accessed February 13, 2012.

Geolytic, Inc. Census CD 1990 long form in 2000 boundaries, Release 1.0 on digital optical disk (CD). 2002b. Available at: http://www.GeoLytics.com. Accessed February 13, 2012.

Geolytic, Inc. Census CD 2000 short form blocks for region 4 AK, AZ, CA, CO, HI, ID, MT, NV, NM, OR, UT, WA and WY, release 1.0 on digital optical disk (CD). 2002c. Available at: http://www.GeoLytics.com. Accessed February 13, 2012.

Geolytics, Inc. Census CD 1980 long form in 2000 boundaries, Release 1.0 on digital optical disk (CD). 2012a. Available at: http://www.GeoLytics.com. Accessed February 13, 2012.

Geolytic, Inc. Summary file 1 2010 in 2000 boundaries on digital optical disk (CD). 2012b. Available at: http://www.GeoLytics.com. Accessed February 13, 2012.

Giovannucci E. An updated review of the epidemiological evidence that cigarette smoking increases risk of colorectal cancer. *Cancer Epidemiology, Biomarkers, and Prevention*. 2001; 10(7):725-731.

Giovannucci E. Epidemiologic studies of folate and colorectal neoplasia: a review. *Journal of Nutrition*. 2002a; 132(Suppl 8):2350S-2355S.

Giovannucci E. Modifiable risk factors for colon cancer. *Gastroenterology Clinics of North America*. 2002b; 31(4):925-943.

Giovannucci E. Diet, body weight, and colorectal cancer: a summary of the epidemiologic evidence. *Journal of Women's Health*. 2003; 12(2):173-182.

Giovannucci E. Alcohol, one-carbon metabolism, and colorectal cancer: recent insights from molecular studies. *Journal of Nutrition*. 2004; 134(9):2475S-2481S.

Giovannucci E. Metabolic syndrom, hyperinsulinemia, and colon cancer: a review. *American Journal of Clinical Nutrition*. 2007; 86(3):s836-s842.

Godley LA, Larson RA. Therapy-related myeloid leukemia. *Seminars in Oncology*. 2008; 35(4):418-429.

González CA, Pera G, Agudo A, Bueno-de-Mesquita HB, Ceroti M, Boeing H, Schulz M, Del Giudice G, Plebani M, Carneiro F, Berrino F, Sacerdote C, Tumino R, Panico S, Berglund G, Simán H, Hallmans G, Stenling R, Martinez C, Dorronsoro M, Barricarte A, Navarro C, Quiros JR, Allen N, Key TJ, Bingham S, Day NE, Linseisen J, Nagel G, Overvad K, Jensen MK, Olsen A, Tjønneland A, Büchner FL, Peeters PH, Numans ME, Clavel-Chapelon F, Boutron-Ruault MC, Roukos D, Trichopoulou A, Psaltopoulou T, Lund E, Casagrande C, Slimani N, Jenab M, Riboli E. Fruit and vegetable intake and the risk of stomach and oesophagus adenocarcinoma in the European Prospective Investigation into Cancer and Nutrition (EPIC-EURGAST). *International Journal of Cancer*. 2006; 118(10);2559-2566.

Goodman MT, Shvetsov YB. Rapidly increasing incidence of papillary serious carcinoma of the peritoneum in the United States: fact or artifact? *International Journal of Cancer*. 2009; 124(9):2231-2235.

Grant DJ, Moorman PG, Akushevich L, Palmieri RT, Bentley RC, Schildkraut JM. Primary peritoneal and ovarian cancers: an epidemiological comparative analysis. *Cancer Causes and Controls*. 2010; 21(7):991-998.

Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. *BMC Public Health*. 2009; 9:88.

Half E, Bercovich D, Rozen P. Familial adenomatous polyposis. *Orphanet Journal of Rare Diseases*. 2009; 4:22.
Hoei-Hansen CE, Rajpert-De Meyts E, Daugaard G, Skakkebaek NE. Carcinoma in situ testis, the progenitor of testicular germ cell tumours: a clinical review. *Annals of Oncology*. 2005; 16(6):863-868.

Huyghe E, Matsuda T, Thonneau P. Increasing incidence of testicular cancer worldwide: a review. *Journal of Urology*. 2003; 170(1):5-11.

International Agency for Research on Cancer (IARC). Home page. World Health Organization: International Agency for Research on Cancer: Lyon, France. 2012. Available at: http://www.iarc.fr/ on March 26, 2012.

Jakszyn P, Gonzalez CA. Nitrosamine and related food intake and gastric and oesophageal cancer risk: a systematic review of the epidemiological evidence. *World Journal of Gastroenterology*. 2006; 12(27):4296-42303.

Jarvholm B, Sanden A. Lung cancer and mesothelioma in the pleura and peritoneum among Swedish insulation workers. *Occupational and Environmental Medicine*. 1998; 55(11):766-770.

Jasperson KW, Tuohy TM, Neklason DW, Burt RW. Hereditary and familial colon cancer. *Gastroenterology*. 2010; 138(6):2044-2058.

Jekel JF, Elmore JG, Katz DL. Epidemiology, biostatistics and preventive medicine. Philadelphia, PA: WB Saunders Co. 1996. ISBN: 0-7216-5258-1.

Kamangar F, Dores GM, Anderson WF. Patterns of cancer incidence, mortality, and prevalence across five continents: defining priorities to reduce cancer disparities in different geographic regions of the world. *Journal of Clinical Oncology*. 2006; 24(14):2137-2150.

Kamangar F, Wong-Ho C, Abnet C, Dawsey S. Environmental causes of esophageal cancer. *Gastroenterology Clinic of North America*. 2009; 38(1):27-57.

King RJB, Robins MW. "1. What is cancer?" *In* King RJB, Robins MW, *eds*. Cancer biology, 3rd Ed., San Francisco, CA.: Benjamin Cummings Publishing Co. Inc. May 2006; 1-8. ISBN: 0-13-129454-7.

Kingsley BS, Schmeichel KL, Rubin CH. An update on cancer cluster activities at the Centers for Disease Control and Prevention. *Environmental Health Perspectives*. 2008; 115(1):165-171.

Kinkade S. Testicular cancer. *American Family Physician*. 1999; 59(9):2539-2544.

Klein RJ, Schoenborn CA. Age adjustment using the 2000 projected U.S. population. *Statistical Notes*. 2001; 20:1-12.

Kollarova H, Machova L, Horakova D, Janoutova G, Janout V. Epidemiology of esophageal cancer – an overview article. *Biomedical Papers of the Medical Faculty of the University of Palacky, Olomouc, Czech Republic*. 2007; 151(1):17-20.

Kratz CP, Mai PL, Greene MH. Familial testicular germ cell tumours. *Best Practices and Research: Clinical Endocrinology and Metabolism*. 2010; 24(3):503-513.

Lacatos PL, Lakatos L. Risk for colorectal cancer in ulcerative colitis: changes, causes and management strategies. *World Journal of Gastroenterology*. 2008; 14(25):3937-3947.

Langeberg W, Contreras J, Hatch M, Kinney G, Sukhan S, Williams G. Cancer Cluster Workgroup: Protocol for investigating cancer clusters in Utah. Salt Lake City, UT: Utah Department of Health. June 2004.

Larsson SC, Wolk A. Obesity and colon and rectal cancer risk: a meta-analysis of prospective studies. *American Journal of Clinical Nutrition*. 2007; 86(3):556-565.

Lawson AB, Williams FLR. 4. Basic methods. *In*. Lawson AB, Williams FLR. An introductory guide to disease mapping. Southern Gate, Chichester, West Sussex, UK: John Wiley & Sons, Ltd. 2001; 41-52.

Layke JC, Lopez PP. Esophageal cancer: a review and update. *American Family Physician*. 2006; 73(12);2187-2194.

Leone G, Fianchi L, Voso MT. Therapy-related myeloid neoplasms. *Current Opinion in Oncology*. 2011; 23(6):672-680.

Leone G, Mele L, Pulsoni A, Equitani F, Pagano L. The incidence of secondary leukemia. *Haematologica*. 1999; 84(10):937-945.

Liang PS, Chen TY, Giovannucci E. Cigarette smoking and colorectal cancer incidence and mortality: systematic review and meta-analysis. *International Journal of Cancer*. 2009; 124(10):2406-2415.

Mack TM. Sarcomas and other malignancies of soft tissue, retroperitoneum, peritoneum, pleura, heart, mediastinum, and spleen. *Cancer*. 1995; 75(Suppl 1):211-244.

Manecksha RP, Fitzpatrick JM. Epidemiology of testicular cancer. *BJU International*. 2009; 104(9 Part B):1329-1333.
Mann CJ. Observation research methods. Research design II: cohort, cross sectional, and case-control studies. *Emergency Medicine Journal*. 2003; 20:54-60.

McElroy JA, Remington PL, Trentham-Dietz A, Robert SA, Newcomb PA. Geocoding addreses from a large population-based study: lessons learned. *Epidemiology*. 2003; 14(4):399-407.

McGlynn KA and Cook MB. Eteiologic factors of testicular germ cell tumors. *Future Oncology*. 2009; 5(9):1389-1402.

McManus DT, Olaru A, Meltzer SJ. Biomarkers of esophageal adenocarcinoma and Barrett's esophagus. *Cancer Research*. 2004; 64(5):1561-1569.

Menczer J, Chetrit A, Barda G, Lubin F, Fishler Y, Altaras M et al. Frequency of BRCA mutations in primary peritoneal carcinoma in Israeli Jewish women. *Gynecologic Oncology*. 2003; 88(1):58–61.

Moghaddam AA, Woodward M, Huxley R. Obesity and risk of colorectal cancer: a meta-analysis of 31 studies with 70,000 events. *Cancer Epidemiology, Biomarkers, and Prevention*. 2007; 16(12):2533-2547.

Morita M, Kumashiro R, Kubo N, Nakashima Y, Yoshida R, Yoshinaga K, Saeki H, Emi Y, Kakeji Y, Sakaguchi Y, Toh Y, Maehara Y. Alcohol drinking, cigarette smoking, and the development of squamous cell carcinoma of the esophagus: epidemiology, clinical findings, and prevention. *International Journal of Clinical Oncology*. 2010; 15(2):126-134.

National Cancer Institute (NCI). SEER Cancer Statistics Review 1975-2008. Lifetime Risk (Percent) of Being Diagnosed with Cancer by Site and Race/Ethnicity: Males, 17 SEER Areas, 2006-2008 (Table 1.15) and Females, 17 SEER Areas, 2006-2008 (Table 1.16). National Institutes of Health; National Cancer Institute: Bethesda, Maryland. 2011a. Available at: http://seer.cancer.gov/csr/1975_2008/results_merged/topic_lifetime_risk_diagnosis.pdf on March 26, 2012.

National Cancer Institute (NCI). SEER Cancer Statistics Review 1975-2008. Lifetime Risk (Percent) of Dying from Cancer by Site and Race/Ethnicity: Males, Total US, 2006-2008 (Table 1.18) and Females, Total US, 2006-2008 (Table 1.19). National Institutes of Health; National Cancer Institute: Bethesda, Maryland. 2011b. Available at:

http://seer.cancer.gov/csr/1975_2008/results_merged/topic_lifetime_risk_death.pdf on March 26, 2012.

National Cancer Institute (NCI). What is cancer? website. National Institutes of Health; National Cancer Institute: Bethesda, Maryland. 2012a. Available at: http://www.cancer.gov/cancertopics/cancerlibrary/what-is-cancer on March 26, 2012.

National Cancer Institute (NCI). What you need to know about cancer web pages. National Institutes of Health; National Cancer Institute: Bethesda, Maryland. 2012b. Available at: http://www.cancer.gov/cancertopics/wyntk/. 2012b on March 26, 2012.

National Cancer Institute (NCI). State Cancer Profiles home page. National Institutes of Health; National Cancer Institute: Bethesda, Maryland. 2012c. Available online at: http://statecancerprofiles.cancer.gov/ on March 26, 2012.

Neugut AI, Jacobson JS, DeVivo I. Epidemiology of colorectal adenomatous polyps. *Cancer Epidemiology, Biomarkers, and Prevention*. 1993; 2(2):159-176.

O'Callaghan A, Mead GM. Testicular carcinoma. *Postgraduate Medical Journal*. 1997; 73(862):481-486.

Oliver MN, Matthews KA, Siadaty M, Hauck FR, Pickle LW. Geographic bias related to geocoding in epidemiologic studies. *International Journal of Health Geographics*. 2005; 4:29.

Parkin DM, Bray F, Ferlay J, Pisani P. Global cancer statistics, 2002. *CA Cancer Journal for Clinicians*. 2005; 55(2):74-108.

Patton KT, Thibodeau GA. *Mosby's Handbook of Anatomy and Physiology*. Saint Louis, MO.: Elsevier Health Sciences Publishing. 2000. ISBN: 978-0-323-01096-2.

Pischon T, Lahmann PH, Boeing H, Friedenreich C, Norat T, Tjønneland A, Halkjaer J, Overvad K, Clavel-Chapelon F, Boutron-Ruault MC, Guernec G, Bergmann MM, Linseisen J, Becker N, Trichopoulou A, Trichopoulos D, Sieri S, Palli D, Tumino R, Vineis P, Panico S, Peeters PH, Bueno-de-Mesquita HB, Boshuizen HC, Van Guelpen B, Palmqvist R, Berglund G, Gonzalez CA, Dorronsoro M, Barricarte A, Navarro C, Martinez C, Quirós JR, Roddam A, Allen N, Bingham S, Khaw KT, Ferrari P, Kaaks R, Slimani N, Riboli E. Body size and risk of colon and rectal cancer in the European Prospective Investigation Into Cancer and Nutrition (EPIC). *Journal of the National Cancer Institute*. 2006; 98(13):920-931.

Rahbari R, Zhang L, Kebebew E. Thyroid cancer gender disparity. *Future Oncology*. 2010; 6(11):1771-1779.

Read TE, Kodner IJ. Colorectal cancer: risk factors and recommendations for early detection. *American Family Physician*. 1999; 59(11);3083-3092.

Riboli E, Norat T. Epidemiologic evidence of the protective effect of fruit and vegetables on cancer risk. *American Journal of Clinical Nutrition*. 2003; 78(Suppl 3):559S-569S.

Rothman KJ, Boice JD, Epidemiologic analysis with a programmable calculator, New Edition. Boston, MA: Epidemiology Resources, Inc. 1982.

Rustgi AK. The genetics of hereditary colon cancer. *Genes and Development*. 2007; 21(20):2525-2538.

Sahai H, Khurshid A. Confidence intervals for the mean of a Poisson distribution: a review. *Biometrical Journal*. 1983; 35:857-867.

Sahai H. Khurshid A. Statistics in epidemiology: methods, techniques and applications. Boca Raton, FL: CRC Press, Inc. 1996.

Schrager S, Potter BE. Diethylstilbestrol exposure. *American Family Physician*. 2004; 69(10):2395-2400.

Selvin S. 1. Measures of risk: rates and probabilities. *In*. Selvin S. Monographs in epidemiology and biostatistics, Vol 25: Statistical analysis of epidemiologic data. Oxford, UK: Oxford University Press. 1996; 3-40. ISBN: 0-19-509760-2.

Shaheen N, Ransohoff DF. Gastroesophageal reflux, barrett esophagus, and esophageal cancer: scientific review. *JAMA*. 2002; 287(15):1972-1981.

Sill H, Olipitz W, Zebisch A, Schulz E, Wolfler A. Therapy-related myeloid neoplasms: pathobiology and clinical characteristics. *British Journal of Pharmacology*. 2011; 162(4):792-805.

Thornton M, ed. Standards for cancer registries, Vol II, data standards and data dictionary, 16th Ed, Version 12.2. Springfield, Ill.: North American Association of Central Cancer Registries, January 2012. Available at: http://www.naaccr.org/StandardsandRegistryOperations/VolumeII.aspx. Accessed March 27, 2012.

Thun MJ, Henley SJ, Calle EE. Tobacco use and cancer: an epidemiologic perspective for geneticists. *Oncogene*. 2002; 21(48):7307-7325.

Thun MJ, Sinks T. Understanding cancer clusters. *CA Cancer Journal for Clinicians*. 2004; 54(5):273-280.

Toh Y, Oki E, Ohgaki K, Sakamoto Y, Ito S, Egashira A, Saeki H, Kakeji Y, Morita M, Sakaguchi Y, Okamura T, Maehara Y. Alcohol drinking, cigarette smoking, and the development of squamous cell carcinoma of the esophagus: molecular mechanisms of carcinogenesis. *International Journal Clinical Oncology*. 2010; 15(2):135-144.

Utah Cancer Registry (UCR). 2012 Cancer Dataset for the Utah Environmental Public Health Tracking Network: Containing public use data records for primary in-situ Utah resident cancers from 1973 to 2009. Electronic data transfer. January 2012. See: http://ucr.utah.edu/.

U.S. Census Bureau (USCB). Appendix A. Geographic terms and concepts in summary file 3: 2000 census population and housing, technical documentation. SF3/14 RV. Washington DC: U.S. Department of Commerce, Biometrics and Statistics Administration. August 2004. Available at: http://www.census.gov/prod/cen2000/doc/sf3.pdf on February 13, 2012.

U.S. Environmental Protection Agency (USEPA). Toxic Release Inventory web page. Use the TRI Explorer query tool. U.S. Environmental Protection Agency: Washington, DC. 2012a. Available at: http://www.epa.gov/tri/ on March 26, 2012.

U.S. Environmental Protection Agency (USEPA) Superfund site information web page. Use the Superfund site information search tool. U.S. Environmental Protection Agency: Washington, DC. 2012b. Available at: http://www.epa.gov/superfund/sites/cursites/ on March 26, 2012.

Vasen HF, van der Meulen-de Jong AE, de Vos Tot Nederveen Cappel WH, Oliveira J; ESMO Guidelines Working Group. Familial colorectal cancer risk: ESMO clinical recommendations. *Annals of Oncology*. 2009; 20(Suppl 4):51-53.

von Rahden BH, Stein HJ, Siewert JR. Barrett's esophagus and Barrett's carcinoma. *Current Oncology Reports. 2003; 5(3):203-209.*

Waller LA, Gotway CA. 2. Analyzing public health data. *In* Waller LA, Gotway CA *eds*. Applied spatial statistics for public health data. Hoboken, NJ: John Wiley & Sons, Inc. 2004; 7-37.

Ward MH, Nuckols JR, Giglierano J, Bonner MR, Wolter C, Airola M, Mix W, Colt JS, Hartge P. Positional accuracy of two methods of geocoding. *Epidemiology*. 2005; 16(4):542-547.

Warner SC, Aldrich TE. The status of cancer cluster investigations undertaken by state health departments. *American Journal of Public Health*. 1988; 78(3):306-307.

Weinberg RA. "2.The nature of cancer." *In* Weinberg RA, *ed*. Biology of cancer. New York, NY.: Garland Science, Taylor & Francis Group, LLC. June 2006; 25-56. ISBN: 0-08-1534076-1.

Wilkins KL, Woodgate RL. Preventing second cancers in cancer survivors. *Oncology Nursing Forum*. 2008; 35(2):E12-E22.

Wolin KY, Carson K, Colditz GA. Obesity and cancer. *Oncologists*. 2010; 15():556-565.

World Health Organization (WHO). International Classification of Diseases for Oncology, 3rd Edition (ICD-O-3) website. 2012. Available at: http://www.who.int/classifications/icd/adaptations/oncology/en/. Accessed February 13, 2012.

Xie J, Itzkowitz SH. Cancer in inflammatory bowel disease. *World Journal of Gastroenterology*. 2008; 14(3):378-389.

Zhang HY, Spechler SJ, Souza RF. Esophageal adenocarcinoma arising in Barrett esophagus. *Cancer Letters*. 2009; 275(2):170-177.

Zimmerman DL, Li J. The effect of local street network characteristics on the positional accuracy of automated geocoding for geographic health studies. *International Journal of Health Geographics*. 2010; 9:10.

Zisman TL, Rubin DT. Colorectal cancer and dysplasia in inflammatory bowel disease. *World Journal of Gastroenterology*. 2008; 14(17):2662-2669.

Figure 1. Location of the study area in northern Davis County, Utah. The study area includes parts of Clinton, Syracuse, West Point and Clearfield, derived from eight census block groups (49.011.125303.4, 49.011.124303.5, 49.011.125304.2, 49.011.125305.1, 49.011.125401.1, 49.011.125501.3, 49.011.125501.4, and 49.011.125503.5). The estimated 2000 census population for this study area is 20,130 persons.

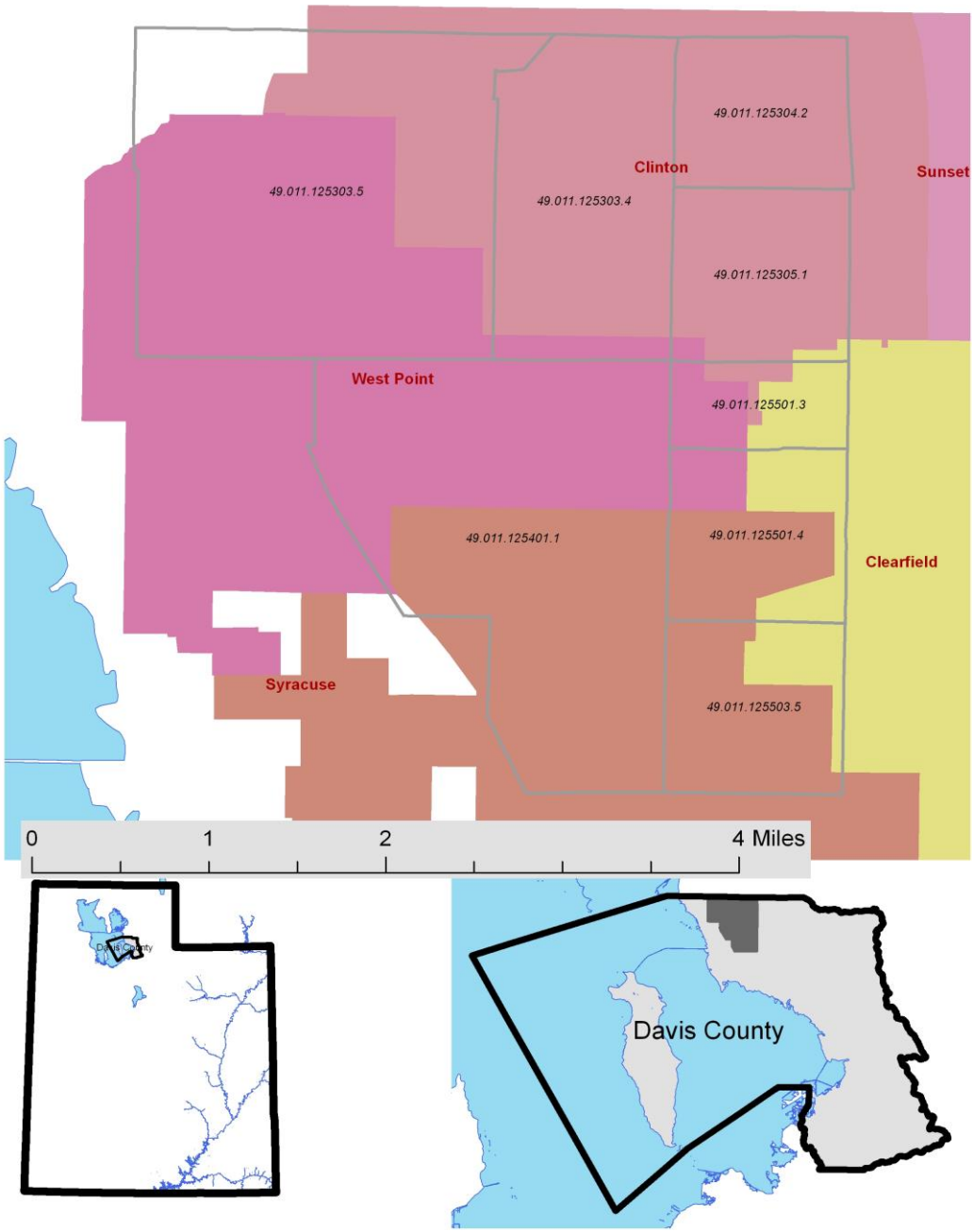

Table 1. Cancer incidence count and indirect age-standardized cancer incidence rate by SEER cancer site category for residents in parts of West Point, Clinton, and Syracuse (Utah 2000 Census Block Groups: 49.011.125303.4, 49.011.124303.5, 49.011.125304.2, 49.011.125305.1, 49.011.125401.1, 49.011.125501.3, 49.011.125501.4, and 49.011.125503.5) from 1975-2009. Cancer categories with sufficient number of cases are further analyzed in 5-year analytical periods. For table brevity, 5-year analytical periods with three or less cases are not evaluated and not reported. The indirect age-standardized cancer incidence rate is per 100,000 person-years. Also shown for comparison is the standardized incidence ratio (and Byar's 95% confidence interval) of cancers for this study area compared to the cancer incidence rates for the state of Utah during the period 1998-2002. Cancer rates that are statistically elevated are annotated with an "S" after the confidence interval.

Cancer Site	Analytical Period	Cancer Incidence Count	Cancer Rate	Standardized Incidence Ratio (and 95% confidence interval)	
Oral cavity and pharynx	1995-1999	6	8.99	1.4 (0.5-3.1)	
	2000-2004	4	4.50	0.7 (0.2-1.9)	
	2005-2009	5	4.34	0.7 (0.2-1.7)	
Esophagus	2005-2009	8	7.71	3.1 (1.3-6.1)	S
Stomach	1990-1994	4	9.33	2.3 (0.6-5.9)	
	2005-2009	4	3.83	1.2 (0.3-3.0)	
Colon	1975-1979	9	38.93	2.2 (1.0-4.3)	S
	1985-1989	9	28.22	1.4 (0.6-2.6)	
	1990-1994	6	14.84	0.7 (0.3-1.5)	
	1995-1999	6	10.90	0.5 (0.2-1.2)	
	2000-2004	20	26.48	1.3 (0.8-2.0)	
	2005-2009	21	20.52	1.1 (0.7-1.6)	
Rectum and recto-sigmoid junction	1995-1999	5	8.32	0.9 (0.3-2.2)	
	2000-2004	8	9.66	1.2 (0.5-2.3)	
	2005-2009	11	9.83	1.2 (0.6-2.2)	

Table 1 (continued). Cancer incidence count and indirect age-standardized cancer incidence rate by SEER cancer site category for residents in parts of West Point, Clinton, and Syracuse (Utah 2000 Census Block Groups: 49.011.125303.4, 49.011.124303.5, 49.011.125304.2, 49.011.125305.1, 49.011.125401.1, 49.011.125501.3, 49.011.125501.4, and 49.011.125503.5) from 1975-2009. Cancer categories with sufficient number of cases are further analyzed in 5-year analytical periods. For table brevity, 5-year analytical periods with three or less cases are not evaluated and not reported. The indirect age-standardized cancer incidence rate is per 100,000 person-years. Also shown for comparison is the standardized incidence ratio (and Byar's 95% confidence interval) of cancers for this study area compared to the cancer incidence rates for the state of Utah during the period 1998-2002. Cancer rates that are statistically elevated are annotated with an "S" after the confidence interval.

Cancer Site	Analytical Period	Cancer Incidence Count	Cancer Rate	Standardized Incidence Ratio (and 95% confidence interval)	
Liver and interhepatic bile duct	2005-2009	5	4.36	1.4 (0.5-3.3)	
Pancreas	1995-1999	4	7.02	1.3 (0.3-3.3)	
	2000-2004	6	8.00	1.2 (0.4-2.7)	
	2005-2009	7	7.11	1.0 (0.4-2.0)	
Other digestive system	2000-2004	4	4.91	3.7 (1.0-9.5)	S
Lung and bronchus	1975-1979	4	16.16	0.9 (0.2-2.3)	
	1980-1984	5	17.30	0.8 (0.3-2.0)	
	1985-1989	5	14.98	0.7 (0.2-1.7)	
	1990-1994	7	16.65	0.7 (0.3-1.5)	
	1995-1999	11	19.53	0.9 (0.4-1.6)	
	2000-2004	17	22.71	1.1 (0.6-1.8)	
	2005-2009	22	22.10	1.1 (0.7-1.6)	

Table 1 (continued). Cancer incidence count and indirect age-standardized cancer incidence rate by SEER cancer site category for residents in parts of West Point, Clinton, and Syracuse (Utah 2000 Census Block Groups: 49.011.125303.4, 49.011.124303.5, 49.011.125304.2, 49.011.125305.1, 49.011.125401.1, 49.011.125501.3, 49.011.125501.4, and 49.011.125503.5) from 1975-2009. Cancer categories with sufficient number of cases are further analyzed in 5-year analytical periods. For table brevity, 5-year analytical periods with three or less cases are not evaluated and not reported. The indirect age-standardized cancer incidence rate is per 100,000 person-years. Also shown for comparison is the standardized incidence ratio (and Byar's 95% confidence interval) of cancers for this study area compared to the cancer incidence rates for the state of Utah during the period 1998-2002. Cancer rates that are statistically elevated are annotated with an "S" after the confidence interval.

Cancer Site	Analytical Period	Cancer Incidence Count	Cancer Rate	Standardized Incidence Ratio (and 95% confidence interval)
Soft tissues (including heart)	2000-2004	5	5.28	2.1 (0.7-4.9)
Cutaneous melanoma	1985-1989	8	16.15	1.4 (0.6-2.9)
	1990-1994	10	16.92	1.3 (0.6-2.3)
	1995-1999	9	12.18	0.8 (0.4-1.5)
	2000-2004	8	8.54	0.5 (0.2-1.0)
	2005-2009	27	22.78	1.0 (0.9-1.4)
Breast	1975-1979	6	44.89	0.7 (0.3-1.6)
	1980-1984	8	49.99	0.8 (0.3-1.5)
	1985-1989	16	82.91	1.0 (0.6-1.7)
	1990-1994	10	41.39	0.5 (0.2-0.9)
	1995-1999	25	76.54	0.9 (0.6-1.3)
	2000-2004	37	85.17	0.9 (0.7-1.3)
	2005-2009	55	96.16	1.2 (0.8-1.4)
Cervix	2000-2004	4	7.69	1.5 (0.4-3.8)

Table 1 (continued). Cancer incidence count and indirect age-standardized cancer incidence rate by SEER cancer site category for residents in parts of West Point, Clinton, and Syracuse (Utah 2000 Census Block Groups: 49.011.125303.4, 49.011.124303.5, 49.011.125304.2, 49.011.125305.1, 49.011.125401.1, 49.011.125501.3, 49.011.125501.4, and 49.011.125503.5) from 1975-2009. Cancer categories with sufficient number of cases are further analyzed in 5-year analytical periods. For table brevity, 5-year analytical periods with three or less cases are not evaluated and not reported. The indirect age-standardized cancer incidence rate is per 100,000 person-years. Also shown for comparison is the standardized incidence ratio (and Byar's 95% confidence interval) of cancers for this study area compared to the cancer incidence rates for the state of Utah during the period 1998-2002. Cancer rates that are statistically elevated are annotated with an "S" after the confidence interval.

Cancer Site	Analytical Period	Cancer Incidence Count	Cancer Rate	Standardized Incidence Ratio (and 95% confidence interval)	
Uterus	1990-1994	7	31.26	1.6 (0.6-3.3)	
	2000-2004	10	23.76	1.3 (0.6-2.5)	
	2005-2009	15	25.96	1.3 (0.7-2.2)	
Ovary	2000-2004	4	9.39	0.9 (0.2-2.3)	
	2005-2009	5	8.89	0.9 (0.3-2.2)	
Prostate	1975-1979	5	46.77	0.8 (0.3-1.9)	
	1980-1984	5	38.82	0.6 (0.2-1.4)	
	1985-1989	7	46.70	0.6 (0.2-1.2)	
	1990-1994	24	124.20	0.9 (0.6-1.4)	
	1995-1999	30	108.30	1.0 (0.7-1.4)	
	2000-2004	42	109.20	0.9 (0.7-1.3)	
	2005-2009	69	130.40	1.1 (0.8-1.4)	
Testis	1990-1994	6	16.89	2.8 (1.0-6.2)	S
	1995-1999	5	11.15	2.0 (0.6-4.7)	
	2000-2004	5	9.12	1.3 (0.4-3.0)	
	2005-2009	8	11.46	1.7 (0.7-3.3)	

Table 1 (continued). Cancer incidence count and indirect age-standardized cancer incidence rate by SEER cancer site category for residents in parts of West Point, Clinton, and Syracuse (Utah 2000 Census Block Groups: 49.011.125303.4, 49.011.124303.5, 49.011.125304.2, 49.011.125305.1, 49.011.125401.1, 49.011.125501.3, 49.011.125501.4, and 49.011.125503.5) from 1975-2009. Cancer categories with sufficient number of cases are further analyzed in 5-year analytical periods. For table brevity, 5-year analytical periods with three or less cases are not evaluated and not reported. The indirect age-standardized cancer incidence rate is per 100,000 person-years. Also shown for comparison is the standardized incidence ratio (and Byar's 95% confidence interval) of cancers for this study area compared to the cancer incidence rates for the state of Utah during the period 1998-2002. Cancer rates that are statistically elevated are annotated with an "S" after the confidence interval.

Cancer Site	Analytical Period	Cancer Incidence Count	Cancer Rate	Standardized Incidence Ratio (and 95% confidence interval)
Bladder	2000-2004	4	5.61	1.0 (0.3-2.5)
	2005-2009	6	12.68	1.3 (0.5-2.9)
Kidney and renal pelvis	2005-2009	11	9.65	1.1 (0.6-2.0)
Brain	1990-1994	7	11.00	2.0 (0.8-4.1)
	1995-1999	8	10.38	1.8 (0.8-3.6)
	2000-2004	5	4.99	0.9 (0.3-2.1)
	2005-2009	5	3.80	0.7 (0.2-1.5)
Thyroid	1985-1989	5	8.83	1.8 (0.6-4.3)
	1995-1999	5	5.93	0.9 (0.3-2.1)
	2000-2004	8	7.56	0.8 (0.4-1.6)
	2005-2009	16	11.86	0.9 (0.5-1.4)

Table 1 (continued). Cancer incidence count and indirect age-standardized cancer incidence rate by SEER cancer site category for residents in parts of West Point, Clinton, and Syracuse (Utah 2000 Census Block Groups: 49.011.125303.4, 49.011.124303.5, 49.011.125304.2, 49.011.125305.1, 49.011.125401.1, 49.011.125501.3, 49.011.125501.4, and 49.011.125503.5) from 1975-2009. Cancer categories with sufficient number of cases are further analyzed in 5-year analytical periods. For table brevity, 5-year analytical periods with three or less cases are not evaluated and not reported. The indirect age-standardized cancer incidence rate is per 100,000 person-years. Also shown for comparison is the standardized incidence ratio (and Byar's 95% confidence interval) of cancers for this study area compared to the cancer incidence rates for the state of Utah during the period 1998-2002. Cancer rates that are statistically elevated are annotated with an "S" after the confidence interval.

Cancer Site	Analytical Period	Cancer Incidence Count	Cancer Rate	Standardized Incidence Ratio (and 95% confidence interval)	
Hodgkin lymphoma	2000-2004	5	4.81	2.3 (0.7-5.3)	
	2005-2009	6	4.49	1.8 (0.7-3.9)	
Non-Hodgkin lymphoma	1980-1984	7	21.77	2.7 (1.1-5.5)	S
	1985-1989	6	15.43	1.5 (0.6-3.3)	
	1995-1999	9	14.01	1.1 (0.5-2.0)	
	2000-2004	9	12.08	0.9 (0.4-1.6)	
	2005-2009	18	16.33	1.1 (0.6-1.7)	
Lymphocytic leukemia	2000-2004	4	4.55	0.9 (0.2-2.2)	
	2005-2009	7	6.21	1.1 (0.4-2.3)	
Other sites/types	1995-1999	7	12.81	2.2 (0.9-4.6)	
	2000-2004	7	9.38	1.2 (0.5-2.4)	
	2005-2009	6	6.27	0.7 (0.3-1.6)	

DEFINITIONS

ACS — American Cancer Society. The ACS is a nationwide privately funded voluntary health organization dedicated to eliminating cancer. It was founded in 1913 in New York City. The ACS funds cancer research, works to establish public policy and health care standards, provides support to cancer victims and distributes information about cancer as part of a public educational and awareness campaign. For more information see: http://www.cancer.org/.

AGRC — Utah Automated Geographic Reference Center. This is a unit of the Utah Department of Technology Services. The AGRC facilitates the use of geographic information system (GIS) tools and data by Utah governmental agencies and the public. The AGRC maintains the Utah State Geographic Information Database (SGID) which includes over 300 GIS layer files. For more information see: http://gis.utah.gov/.

CERCLIS — Comprehensive Environmental Response, Compensation, and Liability Information System. The CERCLIS was established as part of the CERCLA act of 1980 (also called Superfund). It is maintained by the U.S. Environmental Protection Agency (EPA). The purpose of the CERCLIS is to store information about abandoned industrial sites that are contaminated with hazardous substances. CERCLIS is used to rank the risks of releases of hazardous substances and the potential to endanger public health or the environment and to prioritize these sites for clean-up. Sites in the CERCLIS that are approved for clean-up are known as National Priority List (NPL) or "Superfund" sites. For more information see: http://www.epa.gov/superfund/sites/cursites/.

DCDH — Davis County Health Department. Once of twelve local health departments serving Utah residents. For more information see: http://www.co.davis.ut.us/health/default.cfm.

EEP — Environmental Epidemiology Program. A program within the Bureau of Epidemiology, Division of Disease Control and Prevention, UDOH. The EEP was established in 1996 and is responsible for investigating diseases related to the environment. The program has two sections. One section conducts surveillance and data management activities including managing the UEPHTN. The other section conducts health hazards risk assessment, including cancer investigations. The program is staffed by personnel with experience and expertise in environmental epidemiology, environmental sciences, toxicology, statistics, public health informatics and geomatics, and health education. For more information see: http://health.utah.gov/enviroepi/.

GeoLytics — GeoLytics is a commercial vendor of census and demographic data calibrated to the 2000 census boundaries. The EEP has purchased 1970, 1980, 1990, 2000 and 2010 census data from GeoLytics to be the basis for estimating intercensal population counts for each of the 1481 census block group boundaries in Utah. Population counts are aggregated into 5-year age groups for each sex. For more information see: http://www.geolytics.com/

IARC — International Agency for Research on Cancer. The IARC is a standards-setting body of the World Health Organization (WHO) for research into the causes of human cancer, the mechanisms of carcinogensis, and strategies for cancer prevention and control. For more information see: http://www.iarc.fr/. The IARC categorizes cancer-causing agents, mixtures and exposures into five categories:

- Group 1: carcinogenic to humans
- Group 2A: probably carcinogenic to humans, meaning there is limited evidence about the carcinogenic potential for humans and sufficient evidence of carcinogenicity for animals.
- Group 2B: possibly carcinogenic to humans, meaning there is limited evidence about the carcinogenic potential for humans or animals
- Group 3: not classifiable
- Group 4: not carcinogenic to humans

ICD-O-3 — International Classification of Disease - Oncology, 3rd Edition. The ICD-O-3 is one of a number of internationally established coding standards for coding site (topography) and histology (morphology) of neoplasms (cancers). For more information see: http://www.who.int/classifications/icd/adaptations/oncology/en/.

NAACCR — North American Association of Central Cancer Registries. NAACCR was established in 1987 as a collaborative professional organization for cancer registries, governmental agencies and professional associations that work with cancer registries. All central cancer registries in the United States and Canada are members. The purpose of NAACCR is to promote standards and enhance the quality of cancer registry data. The NAACCR also promotes training, epidemiologic research, public health activities and patient care improvement policies related to cancer. For more information see: http://www.naaccr.org/.

NCI — National Cancer Institute. The NCI is one of the National Institutes of Health and part of the U.S. Department of Health and Human Services. The NCI was established under the National Cancer Act of 1937 and is primarily responsible for conducting surveillance and research about cancer incidence, diagnosis, prevention, treatment, and rehabilitation. The SEER program is operated by the NCI. For more information see: http://www.cancer.gov/.

SAS	SAS is a robust internationally-accepted statistical software for conducting data management and analysis. The SAS application provides many statistical methodologies that can be applied to data tables. For more information see: http://www.sas.com/.
SEER	Surveillance, Epidemiology and End Results Program. The SEER program is an agency within the NCI. The SEER program works with state cancer registries to develop and disseminate incidence and mortality statistics about cancer in the United States. The SEER program also establishes standards for the analysis of cancer data and interpretation of cancer statistics. For more information see: http://seer.cancer.gov/.
TRI	Toxic Release Inventory. The Toxic Release Inventory is a database of reported releases of toxic chemical and other waste management activities in the United States. The TRI database is maintained by the U.S. Environmental Protection Agency (EPA). The TRI was established under the Emergency Planning and Community Right-to-Know Act of 1986. The TRI requires active industries to report the amount of certain reportable hazardous materials that were released into the environment. For more information see: http://www.epa.gov/tri/.
UCR	Utah Cancer Registry. The UCR is operated under authority from the UDOH by the University of Utah. The UCR was established in 1966 to be a state-wide population-based cancer registry. Utah administrative rule requires the reporting of cancer diagnoses to the UCR. The UCR collaborates with the NCI, SEER and the North American Association of Central Cancer Registries to implement data standards for cancer data. The UCR provide cancer to the EEP through the UEPHTN. For more information, see: http://ucr.utah.edu/.
UDOH	Utah Department of Health. The UDOH is one of the executive agencies within Utah state government. The UDOH strives to improve health in Utah through promoting healthy lifestyles, evidence-based interventions, creating healthy and safe communities and eliminating health disparities. The EEP is a program within the UDOH. For more information, see: http://health.utah.gov/.
UEPHTN	Utah Environmental Public Health Tracking Network. The UEPHTN is a data warehouse that contains health outcome, environmental and supporting data. Data from the UCR and population data derived from the USCB is warehoused in the UEPHTN. For more information see: http://health.utah.gov/enviroepi/activities/EPHTP/NewEPHT/ephtpnew.htm.
USCB	U.S. Census Bureau. Officially the “Bureau of the Census,” the USCB is an

agency authorized by Federal law, within the U.S. Department of Commerce that is charged with preparing and conducting regular surveys and censuses of the United States population. In addition to the decennial population survey, the USCB conducts a number of other surveys and has recently implemented the ACS. For more information, see: http://www.census.gov/.

WHO An agency of the United Nations that deals with international health concerns and policies. For more information see: http://www.who.int/en/.

Cancer Incidence: The term incidence refers to new cases occurring in a period of time, usually annually. Cancer incidence is the number of new cases that occurred in a year. New cancer cases occur when a diagnosis is made. The 2009 national age-adjusted incidence rate is 4.64 cancer cases per 1,000 population per year. For more information, see: http://www.cancer.gov/statistics/glossary/incidence.

Cancer Prevalence: The term prevalence refers to the number of cases that exist either at a moment in time or during a period of time (e.g., annual, lifetime, etc.). When using this term, the time should be included. The 2009 national lifetime cancer prevalence rate is approximately 414.65 cases of cancer among 1,000 population. Cancer prevalence is the total number of cases that exist. For more information, see: http://www.cancer.gov/statistics/glossary/prevalence.

Cancer Incidence Rate: This is a ratio of the cancer incidence (the number of new cancer diagnoses) over the total population. The computing a multiple year rate, the total population added from each year of the rate period is used to get the rate. For more information, see: http://www.cancer.gov/statistics/glossary/incidence.

Indirect Standardized Incidence Rate. The raw (sometimes called "crude") disease incidence rate (number of case incidences per time period divided by the person-years per period) reflects reality. The raw rate is the simplest and most straightforward summary of the population experience. Interpretation of a disease incidence rate involves a comparison of that rate with some comparison or acceptable rate to determine if the rate in question is high or low. Because rates will almost always involve comparing two populations with two different age distributions, comparison of a raw disease incidence rate with a comparison rate is problematic. It does not make sense to compare the rate of disease of a relatively young population with a relatively older population for a disease that is more common in the elderly and be able to state with confidence that the disease rate is higher or lower than expected. For this reason, when the objective is to compare two rates, age standardized rates are preferable. However, it should be noted that the rate itself, once standardized, is not the exact disease burden. The standardized rate should be of the same magnitude as the raw rate.

The indirect standardization method is the preferable method when the disease count in each age group is small or zero. A disadvantage of the indirect method is that the rate is comparable to the comparison population used in its computation, but is not comparable to other population rates. For example, for this study, the study area disease rates are adjusted using the Utah state population and therefore are comparable to the Utah state rates. However, they are not comparable to the San Juan County rates or to national rates.

The Indirect Standardized Rate for the study area (ISR_M) is calculated by:

$$ISR_M = \frac{C_M}{\sum_{age}\left(\frac{C_{U,age}}{P_{U,age}} P_{M,age}\right)} \times \left(\frac{C_U}{P_U}\right) \times 100{,}000$$

Where:

ISR_M is the Indirect Standardized Rate for the study area.

C_M is the total cancer incidence count for the study area for a specific analytical period (e.g., 1990 - 1994).

$C_{U,age}$ is an age-group (e.g., 0 to 19 year in age, etc.) specific cancer incidence count for the state of Utah for a specific analytical period.

$P_{U,age}$ is the age-group specific count of person-years (e.g., number of 0-19 year olds in 1990 plus number of 0-19 year olds in 1991 plus number of 0-19 year olds in 1992 ..., etc.) for the state of Utah for a specific analytical period.

$P_{M,age}$ is the age-group specific count of person-years for the study area for a specific analytical period.

C_U is the total cancer incidence count for the state of Utah for a specific analytical period.

P_U is the total count of person-years for the state of Utah for a specific analytical period.

For purposes of presentation, it is standard practice to present rates per a population of 100,000 people. For example 60 cases per 100,000 people is easier to understand than 0.00006 cases per person.

E_M is the expected case count of cancer incidence for the study area for a specific analytical period. This is the denominator factor of the first term of the rate formula.

$$E_M = \sum_{age}\left(\frac{C_{U,age}}{P_{U,age}} P_{M,age}\right)$$

Standardized Incidence Ratio. The standardized incidence ratio (SIR) is a way of comparing two rates. When using the indirect standardized rate method, the SIR is the first term of the

$$SIR = \frac{C_M}{\sum_{age}\left(\frac{C_{U,age}}{P_{U,age}} P_{M,age}\right)} = \frac{C_M}{E_M}$$

formula to compute the rate.

$$\overline{\underline{SIR}} = \frac{(C_M + k)}{E_M} \times \left[1 - \left(\frac{1}{3\cdot(C_M + k)}\right) + \left(\frac{\pm 1.96}{3\cdot\sqrt{C_M + k}}\right)\right]^3$$

The Byar 95% confidence limits ($Z_\alpha = 1.96$) can be calculated for the SIR by:

Where: SIR is the standardized incidence ratio. The bar over and under means the upper and lower confidence limits of the SIR.

C_M is the total case count of cancer incidence count for the study area for a specific analytical period.

E_M is the expected case count of cancer incidence for the study area for a specific analytical period.

k is a constant for symmetry. For the upper confidence limit, k = 1. For the lower confidence limit, k = 0.

±1.96 is the normal distribution function for a 95% confidence interval. For the upper confidence interval it is a positive value. For the lower confidence interval it is a negative value.